



EXPRESS MAIL NO.

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicants : Charles L. Magness and Shawn P. Iadonato  
Application No. : 09/707,576  
Filed : November 6, 2000  
For : SYSTEM AND METHOD FOR SELECTIVELY CLASSIFYING  
A POPULATION

Examiner : Anna Skibinsky  
Art Unit : 1631  
Docket No. : 55382-3  
Date : December 16, 2006

Mail Stop Amendment  
Commissioner for Patents  
P.O. Box 1450  
Alexandria, VA 22313-1450

Affidavit of Richard M. Myers, Ph.D. under 37 C.F.R. § 1.132

I, Richard M. Myers, Ph.D. being duly sworn, say:

1. I am an internationally recognized scientist and I am employed as Chairman of the Department of Genetics, Stanford University School of Medicine. I also hold positions as Stanford W. Ascherman Professor of Genetics, and Director, Stanford Human Genome Center, both in the Department of Genetics at Stanford University School of Medicine. I received a Bachelors Degree in Biochemistry in 1977 from the University of Alabama, and a Ph.D. degree in Biochemistry in 1982 from the University of California at Berkeley. I currently serve on the Scientific Advisory Board of Illumigen Biosciences, Inc.

2. I am an author or co-author of over 130 peer-reviewed research articles and I have been invited to give presentations on my research at national and international meetings. I was a founding scientist of Mercator Genetics, Inc., and an original member of the scientific advisory board of Genaissance Pharmaceuticals. I am the recipient of several awards related to my work in genetics and the human genome, including the Wills Foundation Award, 1986-2005; the Pritzker Foundation Award, in 2002; and the Searle Scholar Program, from 1987-1990. These and other awards, as well as the details of my publications, are listed in my curriculum vitae, which is attached as Exhibit 1.

3. On information and belief, claims of the present application have been rejected under 35 U.S.C. § 112, first paragraph. According to the Office Action dated June 16, 2006, claims 1-10, 14-26, 28, 31-44 and 46-55 fail to comply with the enablement requirement because allegedly there is "undue experimentation required to go from the classification results achieved by implementing the invention to drug target identification without some prior knowledge of a relationship between a potential target and a biological condition as claimed." It is my opinion that the methods described in the application taken together with the state of the art at the time of the application support the enablement of the claimed invention. As discussed below, I conclude that undue experimentation would not be required to identify a drug target using the methods of the invention.

4. On information and belief, the Office Action at page 6, lines 3-4, alleges that "there would be an unpredictable amount of experimentation required to practice the claimed invention." Inasmuch as the methods of the present invention relate to epidemiological, statistical, and genetics analyses, I disagree that the amount of "experimentation" is unpredictable. In fact, I disagree with use of the term experimentation as the much of the genetics work (that the examiner appears to believe not enabled in the application) is routine. In my work as a federally funded scientist, like others in my field, I am routinely required to predict the amount of effort required in my proposals to funding agencies. Additionally, over a nine year period, I was a standing

member of two National Institutes of Health sStudy Sections (and Chair of one of them) that review proposals from scientists representing a broad cross-section of skill in these arts. Following that, I was a member for four years of the Advisory Council—the group of experts that give the final sign-off for funding decisions to the Director—of the National Human Genome Research Institute (one of the NIH institutes). In my experience, it is routine for such proposals to make predictions with reasonable accuracy about the amount of effort required for studies similar to those required by the claimed invention.

5. On information and belief, the Office Action at page 10, lines 15-17, alleges that “the method of identifying a drug target based on genetic differences between two groups is not trivial and requires years to complete....” Although I agree that identifying a drug target is not “trivial,” I disagree that it would require years to complete if the claimed methods are followed. Based on my scientific knowledge of molecular genetics and infectious disease, I am familiar with the methods routinely used to identify mutations in genes and to correlate these mutations with protein expression. My own research involves the study of the structure, function and evolution of the human genome, and I design and perform experiments to understand the role of genes in human diseases. In the course of my career spanning over two decades, I have worked closely with numerous scientists of varying levels of training and expertise, and I have collaborated with laboratories of several foreign countries. I also serve as Editor of the publication *Genome Research*, so I am familiar with the skill level of scientists in my areas of expertise. It is my opinion that anyone of ordinary skill in the field of molecular genetics would have a comparable level of familiarity and expertise.

6. I am aware that it is possible and routine to identify a mutation without knowledge of the underlying disease mechanism, and to determine whether that mutation corresponds to phenotypic characteristics of a subpopulation, such as the “at risk unaffected”. For example, I work in the areas of brain and cardiovascular phenotypes as well as infectious diseases, and I study the role of genes in these and other diseases. In these areas and more, it is routine to identify genetic mutations

associated with phenotypic characteristics (e.g. inherited diseases) without previous knowledge of a relationship to the biological condition. In the context of the claim language and the patent specification, I understand that a population "at risk" could be a population exposed to an infectious agent, a subpopulation "at risk and affected" would be a population exhibiting a phenotypic trait, such as evidence of infection, and that a subpopulation "at risk and unaffected" would be a population expected to have the disease on the basis of history of exposure, yet not exhibiting phenotypic evidence of the infection (*i.e.* symptomatic, serum antibodies, *etc.*).

7. As a scientist working closely with physicians and epidemiologists to study the genetic basis of human disease, I am aware of the procedures and requirements for identifying matched individuals for conducting risk analysis of infections and other diseases. In my opinion, identifying "at risk unaffected" and "at risk affected" populations, and obtaining biological samples, such as blood samples, is routine and need not be time-consuming. Such samples are routinely obtained in the course of medical examination and diagnosis. Using well-known methods of sequence analysis and mutation identification, it is entirely feasible and within ordinary skill in this art to identify a genetic difference that correlates with the affected versus unaffected phenotype. Then, using the vast resources available in the gene databases, including sequences obtained as a result of the Human Genome Project, it would not require undue experimentation to identify a protein or regulatory region that correlates with the observed genetic difference. Indeed, I have either led or been actively involved in several studies that identified DNA sequence variants that are associated with human diseases, including an inherited form of childhood epilepsy, hemochromatosis, basal cell nevus syndrome (of which skin cancer is a symptom), and several others. I participated in the Human Genome Project from its inception, and my laboratory was funded to collaborate on the sequencing of human chromosomes 5, 16 and 19. As a result of this work, I am familiar with the use of the human genome sequences and their availability and utility to anyone of ordinary skill in this art.

8. On information and belief, the Office Action at page 7, lines 2-3, alleges that “the claims are broad in that they are drawn to identification of a drug target for any given biological condition.” Based upon my experience and the prior art, it is my belief that the methods of the claimed invention are broadly applicable; certainly applicable beyond simply infectious disease.

9. On information and belief, the Office Action at page 8, line 18, alleges “a polypeptide is not a drug target” as a rationale for not accepting applicant’s prior supportive arguments. I strongly disagree with this position and submit that entire classes of important drugs (e.g. monoamine oxidase inhibitors and angiotensin converting enzyme (ACE) inhibitors) act on polypeptide drug targets. In fact, most small molecule drugs are inhibitors of proteins, thus making the proteins themselves the drug targets. Furthermore, there are well-known polypeptide therapies (e.g. insulin and growth hormone) where a polypeptide is the drug itself.

10. On information and belief, the Office Action at page 10, lines 4-5, alleges that “the details for identifying a drug target based on the classification are not described. Thus, there is not sufficient support to enable one skilled in the art of make or use the invention.” I strongly disagree with this statement for the following reasons. First, applicants do describe general methods to identify functional mutations differentially associated with the ARU and ARA groups. Second, association methods for identifying genetic mutations, such as those described by example in the application, have been well known in the art since the 1990s. Third, identification of such mutations by comparison with the “at risk unaffected” group that de facto identifies a target. This latter point is in contrast to the traditional analysis using only “at risk affected” groups for disease gene identification which does not generally identify a target.

11. It is an advantage of the present methods that no traditional biochemical screening is required. I am well-qualified to provide an opinion on the differences between traditional biochemistry-based screening, and the new methods disclosed and claimed in this patent application. Instead, screening is performed by computer by

comparing the polynucleotides from the "at risk affected" and "at risk unaffected" groups. One of ordinary skill will be familiar with the input of data from human samples and the methods and search parameters for identifying genetic differences between two samples. This genetic, information-based analysis is a far more efficient, fast, and cost-effective method of identifying the relevant biochemical difference or differences between at-risk populations with and without the disease. It is the comparison of the observed genetic difference (e.g. point mutation, deletion, insertion) with the database to pinpoint the modified region and identify the function that leads directly to identification of a target.

12. On information and belief, the Office Action at page 10, lines 14-15, alleges that "identification of a drug target requires the sorting out of 1000's of targets present in most organisms." Based upon my knowledge of genetics and my direct experience in the field over the course of the past two decades, this statement is incongruous with the history of biomedical research.

13. In conclusion, I am a person with knowledge of the ordinary skill in this art, and in my professional capacity I will be a consumer of the new methods provided by the patent application. It is within my ability to understand and follow the claimed methods. For any disease in which the phenotype is related to a genetic difference, I would expect the claimed methods to allow me to discover that difference, and to correlate it with a gene product, such as a protein. That gene product in turn will correlate with the phenotypic difference between "at risk unaffected" (ARU) and "at risk affected" (ARA) individuals. This information will allow me to identify a target around which a treatment could be designed to mimic the protective phenotype of the "at risk unaffected" individuals.

I further declare that all statements made herein of my own knowledge are true and that these statements are made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under § 1001 of Title 18 of the United States Code.

Richard M. Myers  
Richard M. Myers, Ph.D.

State of California )  
San Mateo ) ss.  
County of Santa Clara

On this 16 day of December 2006, before me, a Notary Public in and for the State and County aforesaid, personally appeared Richard M. Myers, ~~Ph.D.~~, to me known and known to me to be the person of that name, who signed and sealed the foregoing instrument, and he acknowledged the same to be his free act and deed.

Orin Dean  
Notary Public

Commission expires Jan 13<sup>th</sup>, 2009

# **Curriculum vitae**

## **Richard M. Myers, Ph.D.**

**TITLE** Stanford W. Ascherman Professor and Chairman, Department of Genetics, and Director, Stanford Human Genome Center, Stanford University School of Medicine

**BIRTHDATE** March 24, 1954

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Myers Laboratory: [www-shgc.stanford.edu/myerslab/](http://www-shgc.stanford.edu/myerslab/)

### **EDUCATION, RESEARCH AND PROFESSIONAL EXPERIENCE**

2002-present Stanford W. Ascherman Professor and Chairman, Department of Genetics and Director, Stanford Human Genome Center, Stanford University School of Medicine

1996-2002 Professor of Genetics and Director, Stanford Human Genome Center, Stanford University School of Medicine

1993 - 1996 Associate Professor of Genetics and Director, Stanford Human Genome Center, Stanford University School of Medicine

1990-1993 Associate Professor of Physiology and Biochemistry and Biophysics, and Director, Human Genome Center, University of California, San Francisco

1986-1990 Assistant Professor of Physiology and Biochemistry and Biophysics, University of California, San Francisco

1982-1985 Postdoctoral Fellow, Department of Biochemistry & Molecular Biology, Harvard University with Dr. Tom Maniatis

1977-1982 Graduate Student, Department of Biochemistry, University of California, Berkeley with Dr. Robert Tjian (awarded Ph.D. in Biochemistry, 1982)



1972-1977 Undergraduate Student, Department of Biology, University of Alabama, Tuscaloosa; research with Dr. Clifford Hand and Dr. John Hardman (awarded B.S. in Biochemistry, 1977)

## RESEARCH INTERESTS

Molecular basis of human inherited diseases and traits, including Huntington disease, Parkinson disease, bipolar disease, atherosclerosis, hypertension and drug response; genome analysis, including genome-scale analysis of *cis*-acting sequences and DNA binding proteins involved in human gene regulation; genomic and genetic analysis of the protocadherin gene family; genomic basis of vertebrate diversity; human population genetics; large-scale genomic and full-length cDNA sequencing and comparative sequence analysis.

## TEACHING

Co-instructor (with Drs. M. Cherry, A. Sidow and G. Sherlock) of Genetics 211 ("Genomics", a course for graduate students), Stanford University School of Medicine, each Winter Quarter, 2002 - present.

Co-instructor (with Dr. R. Simoni) of Genetics 106Q, a course in the logic of biological science for undergraduate sophomores at Stanford University, Winter Quarter, 1999 - present.

Co-instructor (with Drs. James Ford, Greg Barsh and other Stanford faculty) and Co-director of Genetics 202 ("Medical Genetics", a course for first year medical students), Stanford University School of Medicine, each Fall Quarter, 2003 - present.

Director: Stanford Genome Training Program, Stanford University School of Medicine, 1995 - present.

Co-director: Genetics and Developmental Biology Training Program, Stanford University School of Medicine, 2002 - present.

Co-instructor of Human Biology 2A (a course for undergraduates), Stanford University, Fall Quarter 2005 - present.

Co-instructor (with Dr. D. Vollrath) of Genetics 222 ("Method and Logic in Experimental Genetics", a course for graduate students), Stanford University School of Medicine, each Winter Quarter, 1995 - 2002; guest lecturer 2003, 2005.

Guest Lecturer: BMI 234 ("Medical Genomics", a course for graduate students and medical students), Stanford University School of Medicine, Winter Quarter, 2002.

Guest Lecturer: HRP (a course in genetic epidemiology), Stanford University, 2001 - 2005.

Guest Lecturer: Biology 2S (an undergraduate course in biology and bioethics), Stanford University, 2001 - present.

Guest Lecturer: Genetics 208 ("Human Genetics", a course for graduate and medical students), Stanford University School of Medicine, each Spring Quarter, 1999 - 2003.

Director: Genetics Graduate Program, Stanford University School of Medicine, 1993 - 2001.

Guest Lecturer: Course in Genetic Epidemiology, Cold Spring Harbor Laboratories, 2000, 2002.

Co-instructor (with Drs. R. Simoni, D. Siegmund, D. Cox and D. Botstein) of SME2A, B, C (Science, Mathematics and Engineering), a three-quarter course in the principles of science and mathematics for undergraduate non-science majors at Stanford University, 1996 - 1999.

Organizer of the Genome Seminar Series, a three-quarter series for graduate students and postdoctoral fellows in genome science, as part of the Genome Training Program, 1997 - 2000.

Guest Faculty Instructor, CAM Course for first year graduate students, Stanford University, School of Medicine, 1994 and 1995.

Examiner in Qualifying Exams for 65 graduate students at UCSF and Stanford, 1986 - present.

Member of Dissertation Committees for 57 graduate students at UCSF and Stanford University, 1986 - present.

Co-instructor of Biochemistry 210A and 210B (a course in regulation of biological systems for first year graduate students), Department of Biochemistry & Biophysics, UCSF, seven years (1986-1993).

Co-instructor of Physiology 101 (a course in endocrinology and GI physiology for medical students), Department of Physiology, UCSF, four years (1989-1992).

Co-instructor of Advanced Molecular Cloning Course (a three-week laboratory and lecture course), Cold Spring Harbor Laboratory, four years (1988-1991).

Tutor in the Biochemical Sciences, teaching biochemistry and molecular biology to undergraduates, Harvard University; 1982 - 1985.

Director of a six week laboratory course on DNA cloning techniques, Department of Biology, University of Alabama, 1982.

Teaching Assistant, Department of Biochemistry, University of California, Berkeley, 1979 and 1980.

## **PROFESSIONAL ACTIVITIES**

### **Present Memberships:**

Member: Human Genome Reference Consortium, National Human Genome Research Institute, National Institutes of Health. 2006 - present.

Member and Chair: Review Group, Large-scale DNA Sequencing Centers, National Human Genome Research Institute, National Institutes of Health. 2003 - present.

Member: Coordinating Committee for Prioritization of Sequencing Targets, National Human Genome Research Institute, National Institutes of Health. 2003 - present.

Member: Diversity Committee, Stanford University School of Medicine, 2002 - present.

Member: Biotech Advisory Board, Gunn High School, Palo Alto, 2005 - present.

Member and Chair: HapMap Advisory Committee, National Human Genome Research Institute, National Institutes of Health. 2002 - present.

Member: Stanford Genetics/San Jose Tech Museum Science Education Partnership. 2001 - present.

Associate Editor: Genome Research (Cold Spring Harbor Laboratory Press). 1995 - present.

Member: Scientific Advisory Board, Pharmacogenetics Knowledge Base, Stanford University School of Medicine. 2001- present.

Member: Biology and Biotechnology Program Advisory Committee, U.S. Department of Energy. 2001 - present.

#### **Past Memberships:**

Member: Advisory Council, National Human Genome Research Institute, National Institutes of Health. 2003 - 2006.

Member: ENCODE Advisory Committee, National Human Genome Research Institute, National Institutes of Health. 2002 - 2004.

Member and Chair: Industry Liaison Committee, American Society of Human Genetics. 2002 - 2004.

Member: GRASPP (Genome Resources and Sequencing Prioritization Panel), National Human Genome Research Institute, National Institutes of Health. 2001 - 2003.

Member: Web Site Committee, American Society of Human Genetics. 2001 - 2003.

Member: Committee on Functional Genomics, Genetics and Biocomputation, Stanford University School of Medicine, 1999 - 2002.

Member (and Chair 1999 - 2002): Genome Research Review Committee, National Human Genome Research Institute, National Institutes of Health. 1998 - 2002.

Member: Ad hoc Study Section, Sequencing of additional Drosophila Genomes, National Human Genome Research Institute, National Institutes of Health. 2001.

Member: Board of Directors, American Society of Human Genetics. 1997 - 2001.

Member: Safety Committee, Stanford University, 1996 - 2000.

Member: Ad hoc Study Section, Sequencing of the Rat Genome, National Human Genome Research Institute, National Institutes of Health. 2000.

Member: Intellectual Property Rights Committee, The Human Genome Organization. 1996 - 2000.

Editorial Board Member: Human Molecular Genetics (Oxford University Press). 1992 - 2000.

Member: Special Dean's Review Committee, Department of Genetics, Duke University School of Medicine. 2000.

Member: Study Section, National Human Genome Research Institute, National Institutes of Health. 1994 - 1998.

Member: Mouse Genomics and Genetics Subgroup, Preclinical Models for Cancer Working Group, National Cancer Institute. 1997 - 1998.

Member: Committee on Stanford University School of Medicine/UCSF Academic Priorities and Strategies for Collaboration, Stanford University, 1996 - 1999.

Member (and Chair, 1995-1996): Program Committee, American Society of Human Genetics, 1993 - 1996.

Co-organizer (with Dr. C. Robertson): Biotechnology Training Grant Symposium, Stanford University, 1995.

Member: Advisory Board, Program in Molecular and Genetic Medicine, Stanford University School of Medicine, 1995 - 1999.

Member: Radioisotope Committee, Stanford University School of Medicine, 1994 - 1999.

Co-organizer (with Dr. G. Barsh), Genetics Seminar Series, 1995.

Member: Initial Review Group, National Institutes of Health. Four site visits, 1990 - present.

Member: GESTEC Review Committees, National Center for Human Genome Research (Washington University GESTEC; Whitehead Institute GESTEC): 1994 - 1996.

Member: Board of Scientific Counselors, National Center for Human Genome Research Intramural Research Program. 1994 - 1996.

Member: Scientific Advisory Board, Neurogenetics Center, Duke University School of Medicine, Research Triangle Park, NC, 1994 - 1995.

Ad hoc Council member, National Center for Human Genome Research, National Institutes of Health, 1993 and 1995.

Associate Editor: PCR: Methods and Applications (Cold Spring Harbor Laboratory Press). 1991 - 1995.

Meeting Co-Organizer: Genome Mapping and Sequencing Meeting, Cold Spring Harbor Laboratory, three years (1992 - 1994).

Meeting Organizer: Human Chromosome 4 Workshop, Stanford University, 1993.

Co-organizer and Session Chair, "Winding Your Way Through DNA", a joint UCSF-Exploratorium Symposium for the public on understanding the scientific and societal impact of the recombinant DNA revolution. 1992.

Member of 38 University committees, UCSF. 1986 - 1993.

## **AWARDS AND FELLOWSHIPS**

Honorary Doctorate in Human Letters, December 2005 (University of Alabama).

Blount Initiative Award, October 2003 (University of Alabama).

Pritzker Foundation Award, April 2002 (University of Michigan).

Darden Lecture Award, March 2002 (University of Alabama).

Wills Foundation Award, 1986 - 2003 (at UCSF/Stanford).

Searle Scholar, 1987 - 1990 (at UCSF).

Basil O'Connor Starter Scholar Research Award, 1988 (at UCSF).

Leukemia Society of America Senior Postdoctoral Fellowship, 1984 - 1985 (at Harvard).

Damon Runyon-Walter Winchell Cancer Fund Fellowship, 1982 - 1984 (at Harvard).

Honor Students' Society, 1980 - 1981 (at UCB).

Regents Fellowship, 1979 - 1980 (at UCB).

Abraham Rosenberg and Kaiser Fellowships, 1977 - 1978 (at UCB).

Phi Beta Kappa, 1975 (at UA).

## **PATENTS**

U. S. Patent Number 4,946,773, August 7, 1990, "Detection of base pair mismatches using RNAase A", Thomas P. Maniatis and Richard M. Myers.

U. S. Patent, allowed March 2002, "Mutations in the cystatin B gene in Progressive Myoclonus Epilepsy", Richard M. Myers, David R. Cox, Len A. Pennacchio, Anna-Elina Lehesjoki and Albert de la Chapelle.

## **EDUCATIONAL, COMMUNITY SERVICE AND OTHER ACTIVITIES**

Coach: Palo Alto YMCA Boys' Basketball team, 1996 - 2001.

Coach: Palo Alto YMCA Girls' Basketball team, 2000 - 2003.

Coach: Palo Alto YBAL Boys' Baseball team, kindergarten through grade 1, 1995 - 1996.

Coach and organizer: Palo Alto Boys' Baseball league, grade 2 through grade 8, 1997 - 2003.

Presented science lessons and laboratory tours for Bay Area primary, middle and high schools, each year 1995 - present.

Volunteer: L. M. Nixon Elementary School, Palo Alto, 1995 - 2005.

Volunteer: Terman Middle School, Palo Alto, 2005 - present.

Workshop Presenter: Sally Ride Science Festivals, Stanford University, October, 2003 - 2006.

Member: Biotech Advisory Board, Gunn High School Biotechnology Program, Palo Alto, 2005 - present.

Member: Stanford Genetics/San Jose Tech Museum Science Education Partnership. 2001 - present (see <http://genetics.stanford.edu/techmuseum/>).

## REFEREED PUBLICATIONS

1. Hand, C. W. and Myers, R. M. (1976). Arrhenius parameters for the reaction of oxygen atoms with dicyanoacetylene. *J. Physical Chemistry* **80**: 557-558.
2. Hodo, H. G., Murphy, J., Hardman, J. K. and Myers, R. M. (1977). Substrate interactions with the alpha-subunit of the *Escherichia coli* tryptophan synthase. *Arch. Biochem. Biophys.* **181**: 419-427.
3. Rio, D., Robbins, A., Myers, R. and Tjian, R. (1980). Regulation of simian virus 40 early transcription *in vitro* by a purified tumor antigen. *Proc. Natl. Acad. Sci. USA* **77**: 5706-5710.
4. Myers, R. M. and Tjian, R. (1980). Construction and analysis of simian virus 40 origins defective in tumor antigen binding and DNA replication. *Proc. Natl. Acad. Sci. USA* **77**: 6491-6495.
5. Myers, R. M., Rio, D. C., Robbins, A. K., and Tjian, R. (1981). SV40 gene expression is modulated by the cooperative binding of T antigen to DNA. *Cell* **25**: 373-384.
6. Myers, R. M., Kligman, M. and Tjian, R. (1981). Does simian virus 40 T antigen unwind DNA? *J. Biol. Chem.* **256**: 10156-10160.
7. Myers, R. M., Williams, R. C. and Tjian, R. (1981). Oligomeric structure of a simian virus 40 T antigen in free form and bound to DNA. *J. Mol. Biol.* **148**: 347-353.
8. Brock, P. W., Myers, R., Baker, D. C. and Hardman, J. K. (1983). Photoaffinity labeling of the indole sites on the *Escherichia coli* tryptophan synthase  $\alpha$ -subunit. *Arch. Biochem. Biophys.* **220**: 435-443.
9. Fisher, E. F., Feist, P. L., Beaucage, S. L., Myers, R. M., Tjian, R. and Caruthers, M. H. (1984). Interaction of AD2+D2 protein and simian virus 40 large T antigen with the large tumor antigen binding site I. *Biochemistry* **23**: 5938-5944.
10. Jones, K. A., Myers, R. M., and Tjian, R. (1984). Mutational analysis of simian virus 40 large T antigen binding sites. *EMBO J.* **3**: 3247-3255.

11. Myers, R. M., Lumelsky, N., Lerman, L. S. and Maniatis, T. (1985). Detection of single base substitutions in total genomic DNA. *Nature* **313**: 495-498.
12. Myers, R. M., Fischer, S. G., Maniatis, T. and Lerman, L.S. (1985). Modification of the melting properties of duplex DNA by attachment of a GC-rich DNA sequence as determined by denaturing gradient gel electrophoresis. *Nucleic Acids Res.* **13**: 3111-3130.
13. Myers, R. M., Fischer, S. G., Lerman, L. S. and Maniatis, T. (1985). Nearly all single base substitutions in DNA fragments joined to a GC-clamp can be detected by denaturing gradient gel electrophoresis. *Nucleic Acids Res.* **13**: 3131-3146.
14. Myers, R. M., Lerman, L. S. and Maniatis, T. (1985). A general method for saturation mutagenesis of cloned DNA fragments. *Science* **229**: 242-247.
15. Myers, R. M., Larin, Z. and Maniatis, T. (1985). Detection of single base substitutions by ribonuclease cleavage of mismatches in RNA:DNA duplexes. *Science* **230**: 1242-1246.
16. Myers, R. M., Tilly, K. and Maniatis, T. (1986). Fine structure genetic analysis of a beta-globin promoter. *Science* **232**: 613-618.
17. Milton, D. L., Napier, M. L., Myers, R. M. and Hardman, J. K. (1986). *In vitro* mutagenesis and overexpression of the *E. coli trpA* gene and the partial characterization of the resultant tryptophan synthase mutant alpha-subunits. *J. Biol. Chem.* **261**: 16604-16615.
18. Collins, M. and Myers, R. M. (1987). Alterations in DNA helix stability due to base modifications can be evaluated using denaturing gradient gel electrophoresis. *J. Mol. Biol.* **198**: 737-744.
19. Cowie, A. and Myers, R. M. (1988). DNA sequences involved in transcriptional regulation of the mouse beta-globin promoter in murine erythroleukemia cells. *Molec. Cell. Biol.* **8**: 3122-3128.
20. Cox, D. R., Pritchard, C. A., Uglum, E., Casher, D., Kobori, J., and Myers, R. M. (1989). Segregation of the Huntington disease region of human chromosome 4 in a somatic cell hybrid. *Genomics* **4**: 397-407.
21. Sheffield, V. C., Cox, D. R., Lerman, L. S. and Myers, R. M. (1989). Attachment of a 40-base-pair G+C-rich sequence (GC-clamp) to genomic DNA fragments by the polymerase chain reaction results in improved detection of single-base changes. *Proc. Natl. Acad. Sci. USA* **86**: 232-236.
22. Pritchard, C. A., Casher, D., Uglum, E., Cox, D. R., and Myers, R. M. (1989). Isolation and field- inversion gel electrophoresis analysis of DNA markers located close to the Huntington disease gene. *Genomics* **4**: 408-418.
23. Yost, C. S., Lopez, C. D., Prusiner, S. B., Myers, R. M. and Lingappa, V. R. (1990). Non-hydrophobic extracytoplasmic determinant of stop transfer in the prion protein. *Nature* **343**: 669-672.

24. Stuvé, L. L. and Myers, R. M. (1990). A directly repeated sequence in the beta-globin promoter regulates transcription in murine erythroleukemia cells. *Molec. Cell Biol.* **10**: 972-981.
25. Lopez, C. D., Yost, C. S., Prusiner, S. B., Myers, R. M. and Lingappa, V. R. (1990). Unusual topogenic sequence directs prion protein biogenesis. *Science* **248**: 226-229.
26. Brodsky, M. H., Warton, M., Myers, R. M. and Littman, D. R. (1990). Analysis of the site in CD4 that binds to the HIV envelope glycoprotein. *J. Immunol.* **144**: 3078-3086.
27. Pritchard, C. A., Casher, D., Bull, L., Cox, D. R. and Myers, R. M. (1990). A cloned DNA segment from the telomeric region of human chromosome 4p is not detectably rearranged in Huntington disease patients. *Proc. Natl. Acad. Sci. USA.* **87**: 7309-7313.
28. Cox, D. R., Burmeister, M., Price, E. R., Kim, S. and Myers, R. M. (1990). Radiation hybrid mapping: A somatic cell genetic method for constructing high-resolution maps of mammalian chromosomes. *Science* **250**: 245-250.
29. Duyk, G. M., Kim, S., Myers, R. M. and Cox, D. R. (1990). Exon trapping: A genetic screen to identify transcribed sequences in cloned mammalian genomic DNA. *Proc. Natl. Acad. Sci. USA.* **87**: 8995-8999.
30. Burmeister, M., Cox, D. R. and Myers, R. M. (1990). Dinucleotide repeat polymorphism located at D21S120. *Nucleic Acids Res.* **18**: 4969.
31. deLange, T., Shiue, L., Myers, R. M., Cox, D. R., Naylor, S. L., Killery, A. M. and Varmus, H. E. (1990). Structure and variability of human chromosome ends. *Molec. Cell Biol.* **10**: 518-527.
32. Burmeister, M., Cox, D. R. and Myers, R. M. (1991). TaqI RFLP at D21S137. *Nucleic Acids Res.* **19**: 4020.
33. Burmeister, M., diSibio, G., Cox, D. R. and Myers, R. M. (1991). Identification of polymorphisms by genomic denaturing gradient gel electrophoresis: application to the proximal region of human chromosome 21. *Nucleic Acids Res.* **19**: 1475-1481.
34. Burmeister, M., Kim, S., Price, E. P., de Lange, T., Tantravahi, U., Myers, R. M. and Cox, D. R. (1991). A map of the distal region of the long arm of human chromosome 21 constructed by radiation hybrid mapping and pulsed-field gel electrophoresis. *Genomics* **9**: 19-30.
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## **PUBLICATION OF RESEARCH RESULTS IN PUBLIC DATABASES**

1. More than 450 million base pairs of finished human and other species genomic DNA sequences in GenBank, by Fall 2005. These data can also be viewed at [www-shgc.stanford.edu](http://www-shgc.stanford.edu).
2. Finished DNA sequences for more than 20,000 full-length human, mouse, zebrafish and *Xenopus* cDNAs in GenBank, through Fall 2005. These data can also be viewed at [www-shgc.stanford.edu](http://www-shgc.stanford.edu).

## **Biographical Description (Dr. Richard Myers):**

I received my Ph.D. in Biochemistry from the University of California at Berkeley with Robert Tjian and performed postdoctoral work at Harvard University with Tom Maniatis. I served on the faculty at the University of California at San Francisco and moved to Stanford University School of Medicine in 1993, where I am the Stanford W. Ascherman Professor and Chairman of the Department of Genetics and Director of the Stanford Human Genome Center.

My laboratory uses classical and molecular genetics, genomics, cell biological and computational methods to understand the roles that genes play in a wide range of human traits, including diseases, behaviors and other phenotypes.

Our disease studies focus on brain and cardiovascular phenotypes, including Huntington disease, Parkinson disease, bipolar disease, hypertension and atherosclerosis. These studies involve identifying DNA sequence differences between individuals with disease and unaffected individuals in an effort to understand the genetic basis of the disease. We are part of the Pritzker Consortium, a collaboration between four laboratories to identify gene expression differences in brains of individuals with mood disorders (bipolar disease, major depression and schizophrenia) compared to individuals without disease. We study not only how brains differ in disease, but have also learned how the expression of a variety of cellular pathways is different depending on whether there was a prolonged agonal state prior to death.

A special interest in my group is studying the *cis*- and *trans*-acting elements of transcriptional control in humans at the whole genomic level. We became part of the ENCODE project, funded by the NIH, to identify and understand all the functional elements in a selected 1% of the human genome. This pilot project will soon begin scaling to the entire genome. In addition, since the beginning of the Human Genome Project, I have participated in the large-scale mapping and sequencing of the human genome. My group at the Stanford Human Genome Center, funded by the U.S. Department of Energy in collaboration with the Joint Genome Institute in Walnut Creek California, sequenced human chromosomes 5, 16 and 19, comprising more than 11% of the human sequence. We are also actively involved in comparative genomics, sequencing full-length cDNA clones from a variety of species, sequencing genes and genomes of other organisms and using the information to learn about the biology and evolution of humans.

In addition to my research, I participate in a wide variety of teaching, educational outreach, and institutional and national service activities. I teach in several courses in genetics and genomics to undergraduate and graduate students, and I have a special interest in teaching science to non-science majors. My graduate student teaching has also included serving as Director of the Genetics Graduate Program and two large training grant programs at Stanford. I helped established a partnership between the Department of Genetics and the San Jose Tech Museum ("Stanford at the Tech", see <http://genetics.stanford.edu/techmuseum/>), which helps to develop scientific exhibits as well as providing a venue for training graduate students in the art of teaching to the public. In addition, I direct a variety of teaching activities for the local schools, from the primary level through the junior college level, as well as for a number of laygroups; these include lectures, organized tours of his genome center, laboratory exercises, and curriculum development. I am particularly interested in increasing and nurturing diversity in the scientific community, and I am active in several programs involved with under-represented groups at the graduate school level and earlier.

I currently serve on a variety of advisory panels and editorial boards, including the Advisory Council, the HapMap Advisory Committee and the Review Group for Large-scale DNA Sequencing Centers of the National Human Genome Research Institute, as well as the Biology and Biotechnology Program Advisory Committee for the U.S. Department of Energy. I am an editor of Genome Research and review manuscripts for other journals.

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